

The Effects on Novel Object Recognition by Genetic Reduction of *Dyrk1a* to Normal Levels in Otherwise Trisomic Ts65Dn Down Syndrome Mice

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Down syndrome (DS) is caused by the triplication of chromosome 21 (Hsa21) in humans and is the leading genetic cause of intellectual disability. Ts65Dn mice are used as a model of Down syndrome, with about half of the genes in three copies of those triplicated on Hsa21 in individuals with DS. Overexpression of *Dyrk1a*, a gene found to be triplicated in both individuals with DS and Ts65Dn mice, has been linked to learning and memory deficits. Mice are naturally drawn to novel objects. As such, the Novel Object Recognition (NOR) test can be used to determine if Ts65Dn as compared to normal mice are impaired in discriminating novel objects from previously explored objects. In our current study, Ts65Dn mice with two copies of *Dyrk1a* were compared to Ts65Dn and euploid mice using the NOR task. We hypothesize that Ts65Dn, *Dyrk1a*^{+/-} mice would perform as well as euploid mice on the NOR task, given that they both have two copies of *Dyrk1a*. Our preliminary results indicate that a genotype effect between trisomic mice and euploid mice is not observed. Additionally, Ts65Dn control mice and euploid control mice have a higher discrimination ratio than their *Dyrk1a* knockdown counterparts. These results indicate that overexpression of *Dyrk1a* may not be entirely responsible for deficits in learning and memory.